

Amendments to the Claims:

This listing of claims will replace all prior versions, and listings of claims in the application.

Listing of Claims:

1. (Canceled)

2. (Previously Presented) A method according to claim 17 wherein the antagonist of the CB1 receptor is a specific antagonist of the CB1 receptor.

3-11. (Canceled)

12. (Previously Presented) A method according to claim 17 wherein the CB1 receptor is selected from the group consisting of:

a) a protein having an amino acid sequence comprising SEQ ID NO: 1 or a portion of SEQ ID NO:1, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal;

b) a protein having an amino acid sequence comprising SEQ ID NO: 2 or a portion of SEQ ID NO: 2, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal;

c) an allele of the protein having the amino acid sequence of SEQ ID NO:1 or SEQ ID NO: 2, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal;

d) a protein having the amino acid sequence of SEQ ID NO:1 with a Phenylalanine to Leucine substitution at position 200; and/or an Isoleucine to Valine substitution at position 216; and/or a Valine to Alanine substitution at position 246;

e) a protein having the amino acid sequence of SEQ ID NO: 2 with a Phenylalanine to Leucine substitution at position 139; and/or an Isoleucine to Valine substitution at position 155; and/or a Valine to Alanine substitution at position 185; and

f) a protein comprising the amino acid sequences of SEQ ID NO: 3, SEQ ID NO: 4, SEQ ID NO:5, SEQ ID NO: 6, SEQ ID NO: 7, SEQ ID NO: 8 and SEQ ID NO: 9 or amino acid

sequences 80% homologous to these, said protein having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal.

13. (Previously Presented) A method according to claim 17 wherein the CB1 receptor is a protein having a homology at the amino acid level with SEQ ID NO: 1 of at least 45%, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal.

14. (Previously Presented) A method according to the preceding claim 13 wherein the homology is at least 60%.

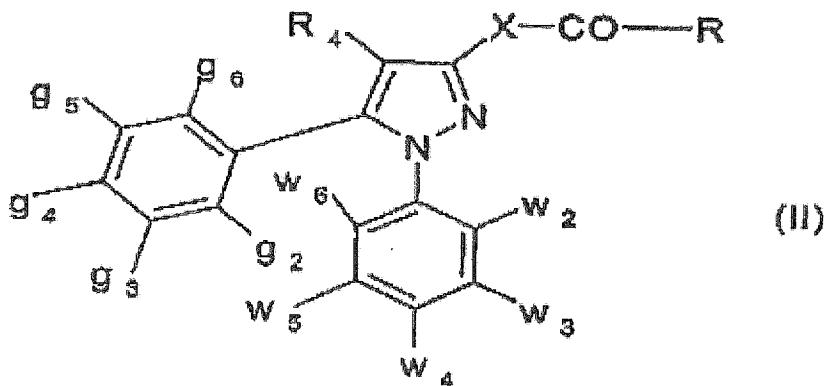
15. (Previously Presented) A method according to claim 17 wherein the daily dosage of CB1 receptor antagonist is from 0.01mg to 500mg.

16. (Canceled)

17. (Currently Amended) A method of treatment of hepatic diseases in a mammal ~~comprising~~consisting essentially of:

administering a therapeutically effective amount of at least one CB1 receptor antagonist to a mammal in need thereof, wherein the CB1 receptor antagonist is a compound of the formula II or one of its pharmaceutically acceptable salt, in which g₂, g₃, g₄, g₅ and g₆ and w₂, w₃, w₄, w₅ and w₆ are identical or different and are independently hydrogen, a chlorine or bromine atom, a (C₁-C₃) alkyl, a (C₁-C₃) alkoxy, a trifluoromethyl or a nitro group and g₄ is optionally a phenyl group; R₄ is hydrogen or a (C₁-C₃) alkyl; X is either a direct bond or a group -(CH₂)_x-N(R₃)-, in which R₃ is hydrogen or a (C₁-C₃) alkyl and x is zero or one; R is: a group -NR₁R₂ in which R₁ and R₂ are independently a (C₁-C₆)-alkyl; an non-aromatic (C₃-C₁₅) carbocyclic radical which is optionally substituted, said substituent (s) being other than a substituted carbonyl; an amino(C₁-C₄) alkyl group in which the amino is optionally disubstituted by a (C₁-C₃) alkyl; a cycloalkyl (C₁-C₃) alkyl in which the cycloalkyl is C₃-C₁₂; a phenyl which is unsubstituted or monosubstituted or polysubstituted by a halogen, by a (C₁-C₅) alkyl or by a (C₁-C) alkoxy; a phenyl (C₁-C₃) alkyl ; a diphenyl (C₁-C₃) alkyl; a naphthyl; an anthracenyl; a saturated 5-to 8-membered heterocyclic radical which is unsubstituted or substituted by a (C₁-C₃) alkyl, by a hydroxyl or by a benzyl; a 1-adamantylmethyl; an aromatic heterocycle which is

unsubstituted or monosubstituted or polysubstituted by a halogen, by a (C₁-C₅) alkyl or by a (C₁-C₅) alkoxy; a (C₁-C₅) alkyl which is substituted by an aromatic heterocycle which is
unsubstituted or monosubstituted or polysubstituted by a halogen, by a (C₁-C₅) alkyl or by a (C₁-C₅) alkoxy; or else R₁ is hydrogen and R₂ is as defined above; or else R₁ and R₂ form a
saturated 5-to 8-membered heterocyclic radical with the nitrogen atom to which they are bonded,
said heterocyclic radical being other than morpholine when w₂, w₃, w₄, w₅, w₆, g₂, g₃, g₄, g₅ and
g₆ are all hydrogen; a group R₂ as defined above when X is -(CH₂)_xN(R₃)-; a group R₅ when X
is a direct bond, R₅ being a (C₁-C₃) alkyl; a (C₃-C₁₂) cycloalkyl which is unsubstituted or
substituted by a (C₁-C₅) alkyl; a phenyl(C₁-C₃) alkyl which is unsubstituted or substituted by a
halogen or by a (C₁-C₅) alkyl; a cycloalkyl (C₁-C₃) alkyl in which the cycloalkyl is C₁-C₁₂ and is
unsubstituted or substituted by a(C₁-C₅) alkyl; or a 2-norbornylmethyl.



18. (Cancelled)

19. (Previously Presented) A method according to claim 17 wherein the CB1 receptor antagonist is N-piperidono-3-pyrazolecarboxamide or one of its pharmaceutically acceptable salt.

20. (Withdrawn) A method according to claim 17 wherein the CB1 receptor antagonist is N-piperidino-5- (4-bromophenyl)-1- (2, 4-dichlorophenyl) -4-ethylpyrazole-3-carboxamide or one of its pharmaceutically acceptable salt.

21. (Previously Presented) A method according to claim 17 wherein the CB1 receptor antagonist is N-piperidino-5- (4-chlorophenyl)-1- (2, 4-dichlorophenyl) -4-methylpyrazole-3-carboxamide or one of its pharmaceutically acceptable salt.

22. (Previously Presented) A method according to claim 17 wherein the hepatic disease is liver fibrosis.

23. (Previously Presented) A method according to claim 17 wherein the hepatic disease is alcoholic liver cirrhosis.

24. (Previously Presented) A method according to claim 17 wherein the hepatic disease is chronic viral hepatitis.

25. (Previously Presented) A method according to claim 17 wherein the hepatic disease is non-alcoholic steatohepatitis.

26. (Previously Presented) A method according to claim 17 wherein the hepatic disease is primary liver cancer.

27. (Previously Presented) A method according to claim 17 wherein the daily dosage of CB1 receptor antagonist is from 1mg to 100mg.